Review article

Air pollution and allergic diseases: Mechanistic pathways, clinical phenotypes, and public health implications

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Abstract

Allergic diseases—including asthma, allergic rhinitis, atopic dermatitis, food allergy, and allergic conjunctivitis—affect ~30–40% of the global population and are rising rapidly. Air pollution ($PM_{2.5}$, PM_{10}, NO_2, O_3, and PAHs) is a major, modifiable environmental driver that contributes to allergic sensitization and worsens disease severity, with disproportionate impacts in children and underserved populations. This narrative review critically synthesizes epidemiological, mechanistic, and clinical evidence linking air pollution to allergic disease, with emphasis on shared mechanisms across organ systems, life-course and gene–environment vulnerability, and implications for prevention and policy.

A structured literature search of PubMed/MEDLINE, Scopus, Web of Science, and Google Scholar identified English-language studies addressing major pollutants, allergic outcomes, and mechanistic pathways. Epidemiological, experimental, clinical, and high-quality reviews were included; non–peer-reviewed and occupational-only reports were excluded. Evidence was synthesized thematically with critical appraisal of relevance and study quality. Across respiratory, cutaneous, ocular, and gastrointestinal phenotypes, air pollution acts as an immune adjuvant via oxidative stress, epithelial barrier dysfunction, and type 2 (Th2) immune polarization mediated by epithelial alarmins (TSLP, IL-33, IL-25). Associations are strongest and most consistent for asthma and allergic rhinitis, with growing coherence for atopic dermatitis; evidence for food allergy and allergic conjunctivitis remains emerging and heterogeneous. Early-life exposure represents a critical window, with gene–environment interactions (e.g., antioxidant and barrier gene variants) contributing to heterogeneity.

Climate change amplifies risk through increased ozone formation, wildfire smoke, extended pollen seasons, and extreme events such as thunderstorm asthma. Pollution-related allergic disease reflects a multisystem “barrier–alarmin–Th2” framework shaped by exposure timing, susceptibility, and climate stressors. While individual mitigation can reduce acute risk, durable prevention requires upstream emission reduction and equity-centered implementation. Future priorities include longitudinal multi-omics, precision prevention, improved exposure assessment, and policy translation that explicitly considers allergic outcomes.

Keywords: Air Pollution, Allergic Sensitization, Asthma, Epithelial Barrier Dysfunction, Type 2 Inflammation

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Background

Allergic diseases—including asthma, allergic rhinitis, atopic dermatitis, food allergy, and allergic conjunctivitis—affect approximately 30–40% of the global population and represent a substantial and growing public health burden (World Health Organization, 2023; Pearce et al., 2007). The prevalence of these conditions has increased markedly over the past several decades, particularly among children and adolescents, suggesting that environmental determinants play a crucial role beyond genetic susceptibility (Asher et al., 2021). Among these environmental factors, ambient air pollution has emerged as one of the most important and modifiable contributors to allergic disease risk. Rapid urbanization, industrial activities, and traffic-related emissions have substantially increased human exposure to pollutants such as particulate matter ($PM_{2.5}$, PM_{10}), nitrogen dioxide (NO_2), sulfur dioxide (SO_2), and ozone (O_3), which are now widely recognized as key drivers of allergic sensitization and disease exacerbation. Globally, air pollution is responsible for millions of premature deaths annually and contributes to a wide spectrum of respiratory and cardiovascular conditions (WHO, 2018). Long-term exposure to particulate matter and gaseous pollutants can penetrate deep into the respiratory tract and circulatory system, triggering oxidative stress, inflammatory responses, and impaired pulmonary function (Lee et al., 2020; Xing et al., 2016). In Southeast Asia and other rapidly developing regions, increasing urbanization, transportation emissions, and biomass burning have further intensified exposure to these pollutants, contributing to rising respiratory and allergic diseases (Pawankar et al., 2020).

A growing body of epidemiological evidence demonstrates strong associations between air pollution exposure and allergic diseases, particularly asthma and allergic rhinitis. Prenatal and early-life exposure to traffic-related pollutants has been linked to increased risk of childhood asthma and allergic sensitization (Gruzieva et al., 2012; Pedersen et al., 2024). Mechanistic studies suggest that pollutants promote allergic inflammation through oxidative stress, epithelial barrier disruption, and activation of type 2 immune pathways, including epithelial-derived cytokines such as thymic stromal lymphopoietin (TSLP), interleukin-33 (IL-33), and interleukin-25 (IL-25) (Miller & Peden, 2014; Lambrecht & Hammad, 2017). Emerging evidence also highlights the roles of microbiome dysbiosis and epigenetic regulation in mediating long-term susceptibility to allergic diseases following environmental exposures (Joubert et al., 2020; Zhang et al., 2023). Beyond respiratory allergic conditions, air pollution has also been associated with dermatologic, ocular, and systemic health outcomes. Epidemiological analyses from Southeast Asia demonstrate that increased concentrations of $PM_{2.5}$ and PM_{10} correlate with higher incidences of respiratory diseases, ocular disorders, and skin conditions (Thongtip et al., 2022). Additionally, climate change and environmental degradation may amplify these effects by increasing airborne allergen production, extending pollen seasons, and intensifying interactions between pollutants and biological allergens (Pawankar et al., 2020). These environmental changes are particularly pronounced in rapidly urbanizing regions of the Asia-Pacific, where increasing industrialization and transportation emissions have contributed to rising rates of allergic diseases.

Despite the growing body of literature linking air pollution to allergic diseases, the evidence remains fragmented across epidemiological studies, mechanistic investigations, and clinical research. Many studies focus primarily on asthma and respiratory allergies, while other allergic conditions—including atopic dermatitis, allergic conjunctivitis, and food allergy—are comparatively underrepresented in integrated analyses. Furthermore, the complex interactions between environmental pollutants, climate change, immune responses, and host susceptibility factors are often examined in isolation rather than within a unified conceptual framework.

Several important knowledge gaps remain in the current literature. First, the association between air pollution and non-respiratory allergic diseases—such as dermatologic and ocular allergic conditions—has not been comprehensively synthesized. Second, mechanistic pathways involving epithelial barrier dysfunction, oxidative stress, microbiome alterations, and immune signaling networks are often studied separately and have rarely been integrated into a coherent pathophysiological model. Third, the combined influence of air pollution and climate-related environmental changes on allergic disease epidemiology

remains insufficiently explored, particularly in rapidly urbanizing regions where exposure levels are increasing. Addressing these gaps is essential for translating emerging scientific evidence into effective clinical management strategies and public health policies aimed at mitigating the health impacts of environmental pollution.

Objectives

This narrative review aims to synthesize current evidence on the relationship between air pollution and allergic diseases by integrating epidemiological, mechanistic, and clinical perspectives. Specifically, this review aims to:

1. Summarize the global epidemiology and burden of allergic diseases associated with air pollution exposure.
2. Examine the biological mechanisms linking air pollution to allergic sensitization and disease progression.
3. Review multisystem clinical manifestations of pollution-related allergic diseases.
4. Discuss implications for prevention strategies, clinical management, and public health policy.

Study Method

This study was conducted as a narrative review with a structured literature search to synthesize current evidence on the relationship between air pollution and allergic diseases. Relevant studies were identified through a systematic search of PubMed/MEDLINE, Scopus, Web of Science, and Google Scholar for articles published between January 2000 and August 2025. Search terms included combinations of keywords related to air pollution and allergic diseases, such as *air pollution, PM2.5, PM10, nitrogen dioxide, ozone, diesel exhaust, wildfire smoke, asthma, allergic rhinitis, atopic dermatitis, food allergy, allergic conjunctivitis, oxidative stress, epithelial barrier dysfunction, Th2 immunity, alarmins, microbiome,* and *epigenetics*. Reference lists of relevant studies and review articles were also screened to identify additional publications.

The study selection process followed the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) framework to ensure transparency. The search identified 425 records, of which 97 duplicates were removed, leaving 328 studies for title and abstract screening. After excluding 219 irrelevant articles, 109 full-text studies were assessed for eligibility. Of these, 49 studies were excluded due to reasons such as occupational-only exposure focus, insufficient methodological detail, or lack of relevance to allergic disease outcomes. Ultimately, 60 studies were included in the qualitative synthesis, as illustrated in the PRISMA flow diagram (Figure 1).

The literature search identified 425 records from four electronic databases and manual reference screening. After removing 97 duplicates, 328 records were screened by title and abstract. Following exclusion of irrelevant studies, 109 full-text articles were assessed for eligibility, and 60 studies were included in the qualitative synthesis of this narrative review.

Studies were eligible if they were peer-reviewed articles published in English examining associations between air pollution exposure and allergic diseases or related biological mechanisms. Eligible studies included epidemiological investigations, cohort studies, experimental and mechanistic research, clinical studies, and high-quality review articles. Findings were synthesized qualitatively, given the heterogeneity in study design, exposure assessment, and outcome definitions. Included studies were critically evaluated based on methodological rigor, relevance, and recency. Potential publication bias and heterogeneity across studies were considered when interpreting the findings.

Results

Clinical Phenotypes and Multisystem Manifestations of Pollution-Related Allergic Disease

Air pollution Air pollution contributes to the development and exacerbation of multiple allergic diseases, including asthma, allergic rhinitis, atopic dermatitis, food allergy, and allergic conjunctivitis. Across organ systems, pollutants act through shared mechanisms—oxidative stress, epithelial barrier disruption, and type 2 (Th2) immune polarization—while interacting synergistically with aeroallergens. Clinical expression is further shaped by exposure timing across the life course, genetic susceptibility, urbanization, socioeconomic inequities, and climate-related events such as wildfires and thunderstorms that intensify exposure (D'Amato et al., 2023; Thien et al., 2018).

Respiratory phenotypes show the strongest and most consistent associations. Exposure to $PM_{2.5}$, NO_2, and diesel exhaust induces oxidative stress and epithelial injury, triggering alarmin release (IL-33, TSLP) and Th2-driven inflammation characterized by IgE production and eosinophilia (Miller & Peden, 2014; Gaffin et al., 2023). Prenatal and early-life exposure represents a critical window of susceptibility, increasing the risk and persistence of childhood asthma (Gruzieva et al., 2019). Climate-driven exposures, particularly wildfire smoke, further elevate exacerbation risk, and pollution-associated severe asthma may exhibit reduced corticosteroid responsiveness, highlighting the therapeutic relevance of upstream biologics such as anti-TSLP agents (Bose et al., 2025). Allergic rhinitis is similarly exacerbated by particulate matter and ozone, which increase nasal epithelial permeability and allergen bioavailability, while ozone-mediated pollen modification enhances allergenicity and contributes to thunderstorm-related outbreaks (Rosario et al., 2024; D'Amato et al., 2023).

Cutaneous and ocular manifestations reflect heightened barrier vulnerability. Atopic dermatitis is worsened by $PM_{2.5}$ and polycyclic aromatic hydrocarbons through oxidative stress, filaggrin downregulation, and epigenetic mechanisms that facilitate allergen penetration and sustained Th2 inflammation, particularly in genetically susceptible children (Fadadu et al., 2023; Soleimani et al., 2020). Allergic conjunctivitis is exacerbated by particulate matter and ozone via oxidative injury and barrier disruption of the ocular surface, with climate-related events such as dust storms and heat waves further increasing exposure in polluted urban settings (Hong et al., 2016; Soleimani et al., 2020). Despite its prevalence, conjunctivitis remains underrepresented in pollution-health research, and socioeconomic barriers limit access to effective treatment (Fadadu et al., 2023).

Evidence linking air pollution to food allergy is emerging but less consistent. Prenatal and early-life exposure to $PM_{2.5}$ and NO_2 may impair oral tolerance by disrupting gut epithelial integrity and microbiota composition, reducing beneficial taxa such as *Bifidobacterium* and promoting alarmin-mediated Th2 sensitization (Zhang et al., 2023; Lambert et al., 2020). Pollutant-induced protein modification may also contribute to cross-sensitization and pollen–food allergy syndromes in urban environments (Chen et al., 2025), although exposure reduction remains the cornerstone of prevention (Holmes et al., 2023).

Overall, air pollution drives multisystem allergic disease through shared immunological and barrier-related pathways while producing organ-specific phenotypes shaped by exposure timing, genetic susceptibility, socioeconomic context, and climate stressors. These interconnected clinical manifestations align with the atopic march framework and provide a foundation for the subsequent Epidemiology and Disease Burden section.

Epidemiology and Disease Burden

The global rise in allergic diseases has been closely linked to increasing air pollution, with the strongest epidemiological evidence for asthma and allergic rhinitis. Asthma affects an estimated 262 million people worldwide and caused approximately 455,000 deaths in 2019, with prevalence ranging from 1% to 18% across countries (WHO, 2023). Childhood asthma prevalence varies substantially, with symptom rates below 4% in parts of Europe, Africa, and India, but exceeding 20% in the UK, Australia, New Zealand, and Brazil (Pearce et al., 2007). Incidence is highest in North America (≈3,200 cases per 100,000 children) and lowest in South Asia (≈356 per 100,000) (Zhao et al., 2024).

In Thailand, asthma remains a major public health concern. Data from the Global Asthma Network Phase I survey in Bangkok reported prevalence rates of 14.6% among children aged 6–7 years and 12.5% among adolescents aged 13–14 years, with severe wheeze affecting 5–7%—slightly higher than the global average of 11% (Global Asthma Network, 2018; Asher et al., 2021).

Epidemiological studies increasingly attribute these patterns to ambient air pollution. In Chengdu, China, a 10 $\mu g/m^3$ increase in $PM_{2.5}$ between 2017 and 2022 was associated with a 2.1% rise in asthma-related hospitalizations, while a similar increase in SO_2 corresponded to a 45.7% increase (Li et al., 2023). Conversely, natural experiments during the COVID-19 pandemic demonstrated rapid health gains following pollution reduction; in Oxford, a 34% decline in $PM_{2.5}$ was accompanied by a 41% reduction in asthma-related emergency admissions (Smith et al., 2024).

Allergic rhinitis is similarly prevalent, affecting approximately 18% of the global population, with marked regional variation influenced by urbanization, environmental exposures, and access to healthcare (Bousquet et al., 2020; Global Allergy Report, 2023). Together, these data underscore the substantial and uneven global burden of allergic diseases and their close association with ambient air pollution (Table 1).

Air Pollution Sources, Mechanisms, and Multisystem Allergic Effects

Air pollution comprises a complex mixture of particulate and gaseous pollutants—including particulate matter (PM), nitrogen dioxide (NO_2), ozone (O_3), sulfur dioxide (SO_2), carbon monoxide (CO), and volatile organic compounds (VOCs)—originating primarily from fossil fuel combustion, traffic, industrial activity, biomass burning, and natural events such as wildfires and dust storms. These exposures contribute substantially to global disease burden, causing an estimated 7–8 million premature deaths annually, with $PM_{2.5}$ alone responsible for approximately 4.2 million outdoor deaths via cardiovascular and respiratory pathways (World Health Organization, 2024). Beyond mortality, air pollution plays a central role in allergic sensitization and disease exacerbation through oxidative stress, epithelial barrier disruption, and immune dysregulation (Reinmuth-Selzle et al., 2017).

Traffic-related pollutants—including NO_2, PM, CO, and secondary ozone—are strongly associated with allergic sensitization, particularly during infancy and early childhood, and are linked to asthma development, lung function impairment, and increased IgE sensitization near roadways (Gruzieva et al., 2012). Industrial emissions, dominated by PM, SO_2, and NO_2, contribute to allergic and respiratory morbidity through overlapping mechanisms but often affect broader geographic regions depending on emission intensity and atmospheric dispersion (Fujitani et al., 2021). While inhalation is the dominant exposure route, dermal and ingestion pathways may be relevant for selected pollutants, with children, older adults, and individuals with pre-existing respiratory disease being particularly vulnerable (Sarica et al., 2023).

The allergenic potential of particulate matter varies by season and geography, reflecting differences in temperature, meteorology, and urban–rural context that modify chemical composition and biological activity (Tables 3–4). Fine and ultrafine particles penetrate deeply into the airways, inducing oxidative stress and epithelial injury, while diesel exhaust particles and ozone chemically modify allergens via oxidation and nitration, increasing IgE-binding capacity and adjuvant activity (Reinmuth-Selzle et al., 2017). Repeated exposure promotes persistence of pollutant–allergen complexes, sustaining immune activation and contributing to bronchial hyperresponsiveness, new sensitizations, and progression along the atopic march (Aljabali et al., 2008; Fowler et al., 2009).

At the mechanistic level, air pollutants act as potent immune adjuvants through interconnected pathways involving oxidative stress, epithelial barrier dysfunction, microbiome disruption, and type 2 (Th2) immune polarization. $PM_{2.5}$ and ozone generate reactive oxygen species, activate redox-sensitive transcription factors (e.g., NF-**K**B, AP-1), and induce epithelial alarmins—TSLP, IL-33, and IL-25—that link innate and adaptive immunity and lower the threshold for Th2-driven inflammation (Li et al., 2023; Valacchi et al., 2020; Fadadu et al., 2023). Barrier disruption facilitates allergen and microbial translocation across the airways, skin, and ocular surface, driving multisystem allergic phenotypes (Patel et al., 2011; Ma et al., 2024).

Pollution also alters airway and gut microbiota, reducing beneficial taxa and impairing immune tolerance, particularly during early life (Mutlu et al., 2018; Marsland & Gollwitzer, 2014). Prenatal and childhood exposures induce epigenetic modifications in immune-regulatory genes, promoting durable Th2 bias and lifelong allergic susceptibility (Gruzieva et al., 2017; Bose et al., 2025). Ultrafine particles exert stronger adjuvant effects than coarse PM, while genetic polymorphisms in antioxidant and barrier-related genes (e.g., *GSTM1*, *FLG*) and age-specific vulnerability explain substantial interindividual variability in allergic outcomes (Bønnelykke et al., 2018; Carlsten et al., 2019).

Climate change further amplifies pollution-related allergic disease by increasing temperatures, altering precipitation, and intensifying extreme events such as

wildfires and thunderstorms, thereby elevating pollutant concentrations and aeroallergen potency (Ziska et al., 2011; Beggs et al., 2022). Thunderstorm asthma and wildfire smoke exemplify this synergy, exposing populations to respirable allergenic fragments and high $PM_{2.5}$ levels that trigger acute exacerbations and may promote new sensitization, particularly in children (D'Amato et al., 2007; Johnston et al., 2014). Urban heat islands further concentrate heat, ozone, and allergens in densely populated areas, disproportionately affecting socioeconomically disadvantaged communities (Guarnieri & Balmes, 2014).

Collectively, air pollution drives heterogeneous allergic phenotypes across the respiratory, cutaneous, ocular, and gastrointestinal systems through shared upstream mechanisms while producing organ-specific effects shaped by particle characteristics, exposure timing, and host susceptibility (Takizawa, 2011). Respiratory outcomes show the strongest causal evidence, while cutaneous, ocular, and food-related phenotypes highlight differential barrier vulnerability and emerging pathways (Rosario et al., 2024; Zhang et al., 2023; Ziou et al., 2023). These findings support the atopic march framework and underscore epithelial protection and early-life exposure reduction as central strategies for prevention.

The key evidence linking major air pollutants with allergic diseases and their proposed biological mechanisms is summarized in Table 5

Therapeutic Interventions and Prevention Strategies

Because air pollution drives allergic disease through oxidative stress, epithelial barrier disruption, and Th2 immune polarization, effective mitigation requires a layered approach combining individual-level protection, environmental control, and upstream exposure reduction. Most current interventions reduce disease severity rather than prevent initial sensitization, with effectiveness influenced by age, genetic susceptibility, and exposure intensity.

Antioxidant supplementation has been investigated given the central role of oxidative stress. While experimental and observational studies suggest potential benefit, randomized clinical evidence remains limited and inconsistent, likely reflecting heterogeneity in nutritional status and genetic background; antioxidants should therefore be considered adjunctive rather than primary therapy (Allan et al., 2010; Moreno-Macías et al., 2014).

Barrier-targeted strategies address early pathways shared across asthma, allergic rhinitis, atopic dermatitis, and food allergy. Proactive skin barrier therapy in infancy and allergen-specific immunotherapy in selected patients show evidence for reducing disease progression (Frew, 2010; Goleva, 2019). Corticosteroids remain foundational for airway disease by suppressing Th2 inflammation and partially restoring epithelial function but do not fully correct barrier vulnerability. Probiotics and regenerative approaches are promising but remain investigational.

Indoor air quality interventions, particularly high-efficiency particulate air (HEPA) filtration, reduce indoor particulate and allergen levels and provide modest improvements in symptoms and bronchial reactivity, especially in children and individuals with asthma (Fisk et al., 2002; Bernstein et al., 2005). These measures complement but cannot substitute for improvements in ambient air quality.

Given substantial interindividual variability, personalized strategies based on sensitization patterns and exposure profiles—including targeted avoidance, exposure monitoring, optimized pharmacotherapy, and immunotherapy selection—are essential to reduce morbidity (Gupta, 2023). Ultimately, sustained prevention depends on reducing pollutant exposure during critical developmental windows through effective air quality regulation and climate mitigation.

Policy and Public Health Strategies for Pollution-Related Allergic Disease

Air pollution is a major, modifiable driver of allergic diseases, particularly asthma and allergic rhinitis, requiring coordinated regulatory action alongside targeted protection for vulnerable populations (WHO, 2021). Emission control policies have demonstrated clear health benefits: reductions in $PM_{2.5}$ following coal and vehicle restrictions in China and enforcement of the U.S. Clean Air Act were associated with improved air quality and fewer asthma-related hospitalizations, although ongoing exceedances highlight the need for sustained enforcement and adaptive urban measures (Leung, 2021; UNEP, 2019; EPA, 2022; Gupta et al., 2018; Canada Health, 2021).

Despite population-level gains, episodic exposures such as wildfires, heatwaves, and traffic surges remain clinically relevant. Evidence-based protective strategies include air quality index monitoring, activity modification, indoor HEPA filtration (30–90% reduction in indoor particulate matter), and use of high-efficiency masks during severe pollution events, tailored to individual risk profiles (Lee et al., 2020; Zhang et al., 2020).

Importantly, benefits are unevenly distributed, with low- and middle-income countries and disadvantaged urban communities experiencing higher exposures and limited access to mitigation resources. Reducing pollution-related allergic disease therefore requires an integrated approach combining upstream emission reduction, downstream protection of high-risk groups, climate mitigation, and equitable implementation to achieve durable public health gains (WHO, 2021).

Discussion

This narrative review integrates epidemiological, mechanistic, and clinical evidence demonstrating that ambient air pollution—particularly $PM_{2.5}$, NO_2, and O_3—is a major and modifiable driver of allergic disease. Associations are strongest and most consistent for asthma and allergic rhinitis, supported by birth-cohort studies and natural experiments showing rapid reductions in asthma morbidity following emission decreases. Evidence for atopic dermatitis is increasingly coherent, whereas links to food allergy and allergic conjunctivitis remain emerging and heterogeneous, highlighting the need for standardized outcomes and longitudinal designs.

A central contribution of this review is the synthesis of shared biological pathways across organ systems into a unified framework. Pollutants act as immune adjuvants through oxidative stress, epithelial barrier disruption, and Th2 immune polarization. Reactive oxygen species generated by $PM_{2.5}$ and O_3 activate redox-sensitive inflammatory pathways, increase epithelial permeability, and promote release of epithelial alarmins (TSLP, IL-33, IL-25), thereby linking inhalational exposure to multisystem IgE-mediated inflammation. This “barrier–alarmin–Th2” axis provides a mechanistic basis for cross-organ sensitization and the atopic march, particularly when exposure occurs during early development.

The evidence further supports microbiome dysbiosis and epigenetic reprogramming as key modifiers of persistence and heterogeneity in allergic risk. Pollution-associated loss of protective microbial taxa and epigenetic alterations in immune-regulatory genes may durably shift immune set points toward pro-allergic responses, consistent with an early-life “biological embedding” hypothesis. Susceptibility is strongly age-dependent and shaped by gene–environment interactions, with prenatal and early childhood representing critical vulnerability windows due to ongoing immune, epithelial, and lung development.

Climate change emerges as a major amplifier of pollution-related allergic disease rather than a parallel driver. Rising temperatures increase ozone formation, intensify wildfire smoke exposure, extend pollen seasons, and enhance allergen potency, contributing to severe exacerbations such as thunderstorm asthma and disproportionately affecting children and socioeconomically disadvantaged communities. These findings underscore the need to integrate climate metrics into allergy research and align air-quality regulation with climate mitigation.

From a prevention and policy perspective, the evidence supports a layered approach combining individual-level exposure reduction with upstream emission control. While measures such as HEPA filtration, activity modification, and protective masks reduce short-term risk, durable prevention depends on protecting early-life windows through sustained air-quality improvement. Mechanistic insights also support the clinical relevance of biologics targeting upstream type 2 pathways for pollution-sensitive phenotypes. Addressing equity is essential, as populations in low- and middle-income countries and disadvantaged urban settings face higher exposures and fewer protective resources.

Finally, important gaps remain. Future research should prioritize longitudinal studies in underrepresented regions, harmonized multi-omics and exposure assessment to identify early biomarkers, and responsible use of AI to integrate high-dimensional data for risk prediction and public health response. Aligning mechanistic insight with precision prevention, equitable implementation, and strong environmental policy offers the most effective path to

reducing the global burden of pollution-driven allergic disease.

Recommendations for Future Research and Policy

Future research should move beyond exposure–outcome associations toward integrated mechanistic insight, precision prevention, and policy translation. As allergic diseases rise with urbanization, climate change, and persistent exposure to PM, NO_2, and O_3, studies should explicitly link biological pathways with clinical relevance and regulatory implications. Longitudinal life-course cohorts using multi-omics approaches—including genomics, epigenomics, microbiomics, and metabolomics—are needed to identify early molecular changes preceding allergic disease. Standardized exposure assessment, harmonized analytical pipelines, and international data sharing will be essential to improve reproducibility and causal inference. Precision prevention should integrate genetic susceptibility, microbiome profiles, and exposure histories to identify high-risk groups. Preventive trials targeting upstream type 2 pathways (e.g., TSLP and IL-33), particularly in pollution-exposed children, warrant priority evaluation. Equity must remain central, with research focused on underserved populations in low- and middle-income countries and disadvantaged urban communities. Emphasis should be placed on scalable, low-cost interventions such as household air filtration and clean cooking technologies. Finally, mechanistic evidence should directly inform air quality standards and public health policy. Incorporating allergic outcomes into regulatory frameworks, supported by AI-based exposure modeling, IoT monitoring, and wearable sensors, can enable adaptive responses to pollution and climate extremes and promote equitable reductions in allergic disease burden.

Conclusion

Air pollution—encompassing $PM_{2.5}$, PM_{10}, NO_2, O_3, and PAHs—is a major driver of allergic sensitization and disease exacerbation through interacting biological pathways that include oxidative stress, epithelial barrier dysfunction, Th2 immune polarization, microbiome dysbiosis, and epigenetic programming. These mechanisms operate across organ systems, linking respiratory, cutaneous, ocular, and food-related allergic phenotypes and contributing to the atopic march. Climate change further amplifies these effects by increasing pollutant loads, allergen potency, and exposure duration.

Clinically, pollution worsens asthma, allergic rhinitis, atopic dermatitis, food allergy, and conjunctivitis, disproportionately affecting children and marginalized populations. While interventions such as air filtration, targeted biologics, and regulatory policies (e.g., the Clean Air Act) mitigate risk, true prevention depends on reducing exposure during critical developmental windows and addressing structural inequities.

Future progress will require integrating multi-omics, precision medicine, and AI-driven tools with equity-centered research and policy innovation. By aligning mechanistic insight with public health action and climate mitigation, it is possible to reduce the global burden of pollution-driven allergic disease and protect the most vulnerable populations.

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References

Allan, K., Kelly, F. J., & Devereux, G. (2010). Antioxidants and allergic disease: A case of too little or too much? *Clinical and Experimental Allergy, 40*(3), 370–380. https://doi.org/10.1111/j.1365-2222.2009.03413.x

American Lung Association. (2023). *Sulfur dioxide*. https://www.lung.org/clean-air/outdoors/what-makes-air-unhealthy/sulfur-dioxide

Basu, R., & Samet, J. M. (2002). Relation between elevated ambient temperature and mortality: A review of the epidemiologic evidence. *Epidemiologic Reviews, 24*(2), 190–202. https://doi.org/10.1093/epirev/mxf007

Breiteneder, H., Peng, Y., Agache, I., Diamant, Z., Eiwegger, T., Fokkens, W. J., Traidl-Hoffmann, C., Nadeau, K., O'Hehir, R. E., O'Mahony, L., Pfaar, O., Torres, M. J., Wang, D., Zhang, L., & Akdis, C. A. (2020). Biomarkers for diagnosis and prediction of therapy responses in allergic diseases and asthma. *Allergy, 75*(12), 3039–3068. https://doi.org/10.1111/all.14582

Breton, C. V., & Marutani, A. N. (2014). Air pollution and epigenetics: Recent findings. *Current Environmental Health Reports, 1*(1), 35–45. https://doi.org/10.1007/s40572-013-0001-9

Brook, R. D., Rajagopalan, S., Pope, C. A., III, Brook, J. R., Bhatnagar, A., Diez-Roux, A. V., Holguin, F., Hong, Y., Luepker, R. V., Mittleman, M. A., Peters, A., Siscovick, D., Smith, S. C., Jr., Whitsel, L., Kaufman, J. D., & American Heart Association Council on Epidemiology and Prevention, Council on the Kidney in Cardiovascular Disease, and Council on Nutrition, Physical Activity and Metabolism. (2010). Particulate matter air pollution and cardiovascular disease: An update to the scientific statement from the American Heart Association. *Circulation, 121*(21), 2331–2378. https://doi.org/10.1161/CIR.0b013e3181dbece1

Brunner, P. M., Guttman-Yassky, E., & Leung, D. Y. M. (2017). The immunology of atopic dermatitis and its reversibility with broad-spectrum and targeted therapies. *Journal of Allergy and Clinical Immunology, 139*(4), S65–S76. https://doi.org/10.1016/j.jaci.2017.01.011

Bumrungrad International Hospital. (2024). *The health risks of PM 2.5*. https://www.bumrungrad.com/en/health-blog/january-2024/the-health-risks-of-pm-2-5

Calderón-Ezquerro, M. C., Guerrero-Guerra, C., & López-Vidal, Y. (2016). Impact of air pollution in respiratory allergic diseases. *Medicina Universitaria, 18*(73), 212–215. https://doi.org/10.1016/j.rmu.2016.10.006

Chen, Z., Zhang, L., Ai, T., Fan, Y., Liu, Y., Wang, L., & Xie, C. (2025). Air pollution and childhood asthma hospitalizations in Chengdu, China: A time-series study. *Journal of Asthma and Allergy, 18*, 229–243. https://doi.org/10.2147/JAA.S498234

D'Amato, G., Annesi-Maesano, I., Urrutia-Pereira, M., Del Giacco, S., Solé, D., Valsecchi, C., ... Cecchi, L. (2023). New developments in climate change, air pollution, pollen allergy, and interaction with SARS-CoV-2. *Atmosphere, 14*(5), Article 848. https://doi.org/10.3390/atmos14050848

Fujitani, Y., Takahashi, K., Saitoh, K., Fushimi, A., Hasegawa, S., Kondo, Y., Tanabe, K., Takami, A., & Kobayashi, S. (2021). Contribution of industrial and traffic emissions to ultrafine, fine, and coarse particles in the vicinity of industrial areas in Japan. *Environmental Advances, 5*, 100101. https://doi.org/10.1016/j.envadv.2021.100101

Glassner, K. L., Abraham, B. P., & Quigley, E. M. M. (2020). The microbiome and inflammatory bowel disease. *Journal of Allergy and Clinical Immunology, 145*(1), 16–27. https://doi.org/10.1016/j.jaci.2019.11.003

Global Climate and Health Alliance. (2025). *Clean air, healthy lives: New roadmap launched to aid governments combatting air pollution.*

Gong, J., Lee, J. S., Lee, Y., Kim, J., & Park, H. (2020). Air pollution, oxidative stress, and human health: A review of epidemiological and molecular studies. *Molecular & Cellular Toxicology, 16*(3), 253–262.

Guarnieri, M., & Balmes, J. R. (2014). Outdoor air pollution and asthma. *The Lancet, 383*(9928), 1581–1592.

Guo, Y., et al. (2024). The role of Nrf2 in the pathogenesis of allergic diseases. *Journal of Allergy and Clinical Immunology, 153*(2), 375–385. https://doi.org/10.1016/j.jaci.2023.09.009

Gruzieva, O., Bellander, T., Eneroth, K., Kull, I., Melén, E., Nordling, E., van Hage, M., Wickman, M., Moskalenko, V., Hulchiy, O., & Pershagen, G. (2012). Traffic-related air pollution and development of allergic sensitization in children during the first 8 years of life. *Journal of Allergy and Clinical Immunology, 129*(1), 240–246. https://doi.org/10.1016/j.jaci.2011.11.001

Hao, Y., Yang, Y., Zhao, H., Chen, Y., Zuo, T., Zhang, Y., Yu, H., Cui, L., & Song, X. (2025). Multi-omics in allergic rhinitis: Mechanism dissection and precision medicine. *Clinical Reviews in Allergy & Immunology, 68*(1). https://doi.org/10.1007/s12016-025-09028-3

Hew, M., Lee, J., Susanto, N. H., Prasad, S., Bardin, P. G., Barnes, S., Ruane, L., Southcott, A. M., Gillman, A., Young, A., Rangamuwa, K., O'Hehir, R. E., McDonald, C., Sutherland, M., Conron, M., Matthews, S., Harun, N., Lachapelle, P., Douglass, J. A., & Irving, L. (2019). The 2016 Melbourne thunderstorm asthma epidemic: Risk factors for severe attacks requiring hospital admission. *Allergy, 74*(1), 122–130. https://doi.org/10.1111/all.13609

Holgate, S. T., Wenzel, S., Postma, D. S., Weiss, S. T., Renz, H., & Sly, P. D. (2015). Asthma. *Nature Reviews Disease Primers, 1*, 15025. https://doi.org/10.1038/nrdp.2015.25

Hong, J., Zhong, T., Li, H., Xu, J., Ye, X., Mu, Z., ... Xu, J. (2016). Ambient air pollution, weather changes, and outpatient visits for allergic conjunctivitis: A retrospective registry study. *Scientific Reports, 6*, 23858. https://doi.org/10.1038/srep23858

Joubert, A. I., Geppert, M., Johnson, L., Mills-Goodlet, R., Michelini, S., Korotchenko, E., Duschl, A., Weiss, R., Horejs-Höck, J., & Himly, M. (2020). Mechanisms of particles in sensitization, effector function and therapy of allergic disease. *Frontiers in Immunology, 11*, Article 133. https://doi.org/10.3389/fimmu.2020.0133

Kauffmann, F., & Demenais, F. (2012). Gene–environment interactions in asthma and allergic diseases: Challenges and perspectives. *Journal of Allergy and Clinical Immunology, 130*(6), 1229–1240. https://doi.org/10.1016/j.jaci.2012.10.038

Kerschbaumer, A., Stern, R., & Lutz, M. (2008). Origin and influence of PM_{10} concentrations in urban and rural environments. In C. Borrego & A.-L. Norman (Eds.), *Air pollution modeling and its application XIX* (pp. 72–80). Springer.

Kuriakose, J. S., & Miller, R. L. (2010). Environmental epigenetics and allergic diseases: Recent advances. *Clinical & Experimental Allergy, 40*(11), 1602–1610. https://doi.org/10.1111/j.1365-2222.2010.03599.x

Lambert, K. A., Lodge, C., Lowe, A. J., Erbas, B., & Dharmage, S. C. (2020). Air pollution and the development of food allergy: A systematic review. *Allergy, 75*(7), 1662–1674. https://doi.org/10.1111/all.14261

Lu, C., Norbäck, D., Li, Y., & Deng, Q. (2020). Early-life exposure to air pollution and childhood allergic diseases: An update on the link and its implications. *Expert Review of Clinical Immunology, 16*(8), 813–827. https://doi.org/10.1080/1744666X.2020.1804868

Lu, H.-F., Zhou, Y.-C., Yang, L.-T., Zhou, Q., Wang, X.-J., Qiu, S.-Q., Cheng, B.-H., & Zeng, X.-H. (2024). Involvement and repair of epithelial barrier dysfunction in allergic diseases. *Frontiers in Immunology, 15*, Article 1348272. https://doi.org/10.3389/fimmu.2024.1348272

McMahon, K., & Launico, M. V. (2025). Carbon monoxide toxicity. In *StatPearls*. StatPearls Publishing. https://www.ncbi.nlm.nih.gov/books/NBK430740/

Mijač, S., Banić, I., Genc, A.-M., Lipej, M., & Turkalj, M. (2024). The effects of environmental exposure on epigenetic modifications in allergic diseases. *Medicina, 60*(1), 110. https://doi.org/10.3390/medicina60010110

Morianos, I., & Semitekolou, M. (2020). Dendritic cells: Critical regulators of allergic asthma. *International Journal of Molecular Sciences, 21*(21), 7930. https://doi.org/10.3390/ijms21217930

Mukharesh, L., Phipatanakul, W., & Gaffin, J. M. (2023). Air pollution and childhood asthma. *Current Opinion in Allergy and Clinical Immunology, 23*(2), 100–110. https://doi.org/10.1097/ACI.0000000000000881

Pawankar, R., Wang, J.-Y., Wang, I.-J., Thien, F., Chang, Y.-S., Abdul Latiff, A. H., Fujisawa, T., Zhang, L., Thong, B. Y.-H., Chatchatee, P., Leung, T. F., Kamchaisatian, W., Rengganis, I., Yoon, H. J., Munkhbayarlakh, S., Recto, M. T., Neo, A. G. E., Pham, D. L., Lan, L. T. T., … Oh, J. W. (2020). Asia Pacific Association of Allergy Asthma and Clinical Immunology white paper 2020 on climate change, air pollution, and biodiversity in Asia-Pacific and impact on allergic diseases. *Asia Pacific Allergy, 10*(1), e11. https://doi.org/10.5415/apallergy.2020.10.e11

Tontini, C., Radzikowska, U., Aranda, C. J., Tynecka, M., Delgado, I., Eljaszewicz, A., Cornejo-García, J. A., Villaseñor, A., Ariza, A., Contreras, N., López-Rodríguez, J. C., Karaguzel, D., Lozano-Ojalvo, D., Castagnoli, R., Eguiluz-Gracia, I., Sokolowska, M., Mayorga, C., Hilger, C., Mortz, C. G., … Pfaar, O. (2025). The use of omics sciences in allergic diseases from bench to bedside: An EAACI position paper. *Allergy, 80*(9), 2442–2483. https://doi.org/10.1111/all.16638

Thongtip, S., Srivichai, P., Chaitiang, N., & Tantrakarnapa, K. (2022). The influence of air pollution on disease and related health problems in Northern Thailand. *Sains Malaysiana, 51*(7), 1993–2002. https://doi.org/10.17576/jsm-2022-5107-04

Weidinger, S., Beck, L. A., Bieber, T., Kabashima, K., & Irvine, A. D. (2018). Atopic dermatitis. *Nature Reviews Disease Primers, 4*, Article 1. https://doi.org/10.1038/s41572-018-0001-z

Wikipedia contributors. (2023). *Asthma*. Wikipedia. https://en.wikipedia.org/wiki/Asthma

World Health Organization. (2021). *Air pollution*. https://www.who.int/health-topics/air-pollution

World Health Organization. (2023). *Asthma*. https://www.who.int/news-room/fact-sheets/detail/asthma

World Health Organization. (2025). *Updated road map for an enhanced global response to the adverse health effects of air pollution*. World Health Organization.

Zhang, X., Lu, C., Li, Y., Norbäck, D., Murthy, P., Sram, R. J., & Deng, Q. (2023). Early-life exposure to air pollution associated with food allergy in children: Implications for “one allergy” concept. *Environmental Research, 216*(Part 1), 114713. https://doi.org/10.1016/j.envres.2022.114713

Table 1 Trends in Allergic Rhinitis Prevalence Across Continents

Continent	Approximate Prevalence of Allergic Rhinitis (%)	Factors Influencing Prevalence	Air Pollution / Atmospheric Conditions	In-text Citation
Global Average	10% to 30% of the population, ~39.7% of children globally	Urbanization, Environmental Exposure, Air pollution	High levels of PM2.5	(Bousquet et al., 2020), (WHO, 2025)
Africa	18.0%	Environmental Exposure Factors, and Urbanization	Increasing urban air pollution, dust, biomass burning	(Akinbami et al., 2023)
Latin America	17.3%	Pollution, Urbanization	High levels of PM2.5 and NO2 in urban areas	(Sanchez et al., 2022)
Asia	8.7% - 24.1%	Air pollution, rapid urbanization	Severe air pollution, especially PM2.5 and O3	(Wang et al., 2021)
Europe	15-25%	environmental and lifestyle changes, urban air pollution	Traffic-related pollution and industrial emissions	(Schmidt et al. 2022)
North America	20-30%	environmental factors, healthcare access, urbanization	Urban smog, ozone, PM from traffic pollution and industrial emissions	(Smith & Johnson, 2020; CDC 2019)

Table 2 Key Air Pollutants, Sources, Health Effects, and Allergic Sensitization Links

Air Pollutant	Major Sources	Key Health & Allergic Effects	Reference(s)
$PM_{2.5}$	Vehicle exhaust, power plants, industrial combustion, construction dust, smoking, indoor cooking; natural dust storms, volcanoes	Penetrates deep airways (“allergen carrier”); induces oxidative stress; exacerbates asthma, COPD; increases cardiovascular risk, lung cancer, and mortality	Bumrungrad International Hospital (2024)
PM_{10}	Combustion-derived particles, fertilizers (sulfates, nitrates), mineral dust, pollen, mold, sea spray	Upper airway irritation; cough, nasal congestion; worsens asthma and rhinitis	Kerschbaumer et al. (2008)
NO_2	Traffic emissions, high-temperature combustion, power generation	Aggravates asthma; promotes airway inflammation; associated with cardiovascular and metabolic disease	Harrison (2006); Ritz et al. (2019)
O_3	Secondary pollutant from photochemical reactions involving NO_2 and sunlight	Irritates eyes and airways; increases asthma symptoms and respiratory morbidity	Harrison (2006)

Table 2 Key Air Pollutants, Sources, Health Effects, and Allergic Sensitization Links (Continued)

Air Pollutant	Major Sources	Key Health & Allergic Effects	Reference(s)
SO_2	Fossil fuel combustion, industrial emissions, volcanic activity	Bronchoconstriction, wheeze, chest tightness; reduced lung function with chronic exposure	American Lung Association (2023)
CO	Incomplete combustion from vehicles, cooking/heating appliances, generators (poor ventilation)	Systemic hypoxia; neurological and cardiovascular toxicity; indirect respiratory risk	McMahon & Launico (2025)
PAHs / VOCs	Combustion processes, solvents, traffic emissions	AhR-mediated immune modulation; promotes Th2 polarization and allergic sensitization	Miller & Peden (2014)
Second-hand Smoke (SHS)	Tobacco products	Exacerbates asthma; alters respiratory microbiome; increases childhood food allergen sensitization	—

Table 3 Impacts of seasonal variations on particulate composition and allergenicity

Seasonal Factor	Impact on Particulate Composition	Impact on Allergenicity	Reference
Higher temperatures	Increased formation of secondary aerosols and vapor-phase pollutants	In combination with high humidity, enhances pollen, mold, and dust mite allergenicity	(Kida & Ziembowicz, 2025)
Winter season	Increased PM from fossil fuel combustion and residential heating; temperature inversions, weak winds, and reduced sunlight promote pollutant accumulation	Longer indoor stays increase exposure to indoor allergens such as dust mites, pet dander, and mold	(Kida & Ziembowicz, 2025)
Summer season	Improved dispersion of pollutants due to stronger air circulation	Warm, humid conditions promote indoor allergen growth; elevated CO_2 increases pollen production	(Kida & Ziembowicz, 2025)
Spring season	Increased PM from biomass burning, agricultural activities, and pollen; rainfall and wind may partially reduce PM	Peak pollen release increases prevalence and severity of allergic rhinitis symptoms	(Kida & Ziembowicz, 2025)
Rainfall events	Wet deposition reduces airborne PM but increases bioaerosols	Promotes mold growth, exacerbating allergic symptoms	(Baesso Moura et al., 2024)

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Table 4 Impacts of geographic variations on particulate composition and allergenicity

Geographic Factor	Impact on Particulate Composition	Impact on Allergenicity	Reference
Urban areas	Higher pollutant concentrations due to dense traffic and industrial activity	Enhanced allergenicity as particulate matter acts as a carrier ("trojan horse") for allergens	(Dėdelė et al., 2019)
Topography	Mountainous terrain promotes pollutant accumulation through reduced dispersion	Lower pollen counts than flat regions, potentially moderating allergen exposure	(Dėdelė et al., 2019)
Proximity to emission sources	Elevated local pollutant levels near emission points (e.g., roads, factories)	Increased allergen sensitization due to higher PM–allergen interactions	(Dėdelė et al., 2019)
Vegetation-dense areas	Leaf surfaces capture and retain particulate matter, reducing airborne PM	Higher pollen exposure despite reduced particulate levels	(Burbank et al., 2025)

Table 5 Summary of Evidence Linking Air Pollution to Allergic Diseases

Air Pollutant	Allergic Disease / Outcome	Key Epidemiological Findings	Proposed Biological Mechanisms	Key References
$PM_{2.5}$ (fine particulate matter)	Asthma	Strong association with increased asthma incidence, exacerbations, and hospitalizations, particularly in children and urban populations. Prenatal and early-life exposure increases childhood asthma risk.	Penetration into lower airways, oxidative stress, epithelial barrier disruption, activation of Th2 immune response, increased cytokines (TSLP, IL-33, IL-25).	Gruzieva et al., 2012; Miller & Peden, 2014; Pedersen et al., 2024
PM_{10} (coarse particulate matter)	Asthma, allergic rhinitis	Higher ambient PM_{10} levels are associated with increased respiratory symptoms, asthma exacerbations, and allergic airway inflammation.	Induces airway inflammation, stimulates immune activation, increases production of reactive oxygen species (ROS).	Xing et al., 2016; Lee et al., 2020
Nitrogen dioxide (NO_2)	Asthma, allergic sensitization	Exposure to traffic-related NO_2 increases asthma prevalence and airway hypersensitivity, especially in urban environments.	Enhances airway inflammation, increases allergen sensitization, impairs epithelial barrier integrity.	Gruzieva et al., 2012
Ozone (O_3)	Asthma exacerbation	Associated with increased emergency visits for asthma and respiratory symptoms globally.	Oxidative airway injury, epithelial damage, increased inflammatory mediator release.	Pawankar et al., 2020
Sulfur dioxide (SO_2)	Allergic airway diseases	Exposure linked to airway irritation and worsening respiratory allergic symptoms.	Bronchoconstriction and inflammatory airway responses.	WHO, 2018

Table 5 Summary of Evidence Linking Air Pollution to Allergic Diseases (Continued)

Air Pollutant	Allergic Disease / Outcome	Key Epidemiological Findings	Proposed Biological Mechanisms	Key References
Air pollution mixture (PM, NO_2, O_3)	Allergic rhinitis	Long-term exposure associated with increased allergic rhinitis prevalence in urban populations.	Mucosal inflammation, immune dysregulation, increased allergen sensitivity.	Pawankar et al., 2020
Air pollution mixture	Atopic dermatitis	Emerging evidence suggests associations between air pollution exposure and increased risk of atopic dermatitis flares.	Skin barrier disruption, oxidative stress, inflammatory cytokine activation.	Zhang et al., 2023
Air pollution mixture	Allergic conjunctivitis	Exposure to airborne pollutants linked to ocular irritation and allergic conjunctivitis symptoms.	Ocular surface inflammation and immune activation.	Thongtip et al., 2022
Air pollution + climate change interaction	Multiple allergic diseases	Climate change increases pollen production, allergen potency, and exposure duration, amplifying allergic disease burden.	Synergistic effects between pollutants and aeroallergens, immune sensitization.	Pawankar et al., 2020

Table 6 Evidence Linking Air Pollution to Allergic Outcomes Across the Life Course

Life Stage	Primary Pollutants	Main Clinical Outcomes	Association with IgE Sensitization	Key Methodological Challenges	Reference
Prenatal & Early Life	Prenatal NO_2, $PM_{2.5}$, PM_{10}, PAHs	↑ cord blood IgE, epigenetic reprogramming, increased childhood atopy	Stronger prenatal exposure associated with altered mucosal immunity and higher asthma/allergy risk	Confounding (SES, maternal smoking); exposure assessment (LUR vs. personal monitoring)	Pedersen et al., 2023
Childhood	Traffic-related air pollution (BC, NO_2, $PM_{2.5}$), O_3, diesel exhaust particles (DEPs)	↑ incidence of asthma, allergic rhinitis, aeroallergen sensitization	Inconsistent; stronger evidence for clinical phenotypes than IgE markers	Outcome definition (self-report vs. objective); heterogeneity in study design and populations	Pedersen M. et al., 2024
Adulthood	DEPs, O_3, PM metrics, NO_2, wildfire smoke	↑ allergic rhinitis burden, disease exacerbations, food allergy persistence	Limited evidence for de novo sensitization; data mainly from occupational studies	Confounding by cumulative exposure; difficulty isolating new sensitization events	Shusterman, 2024

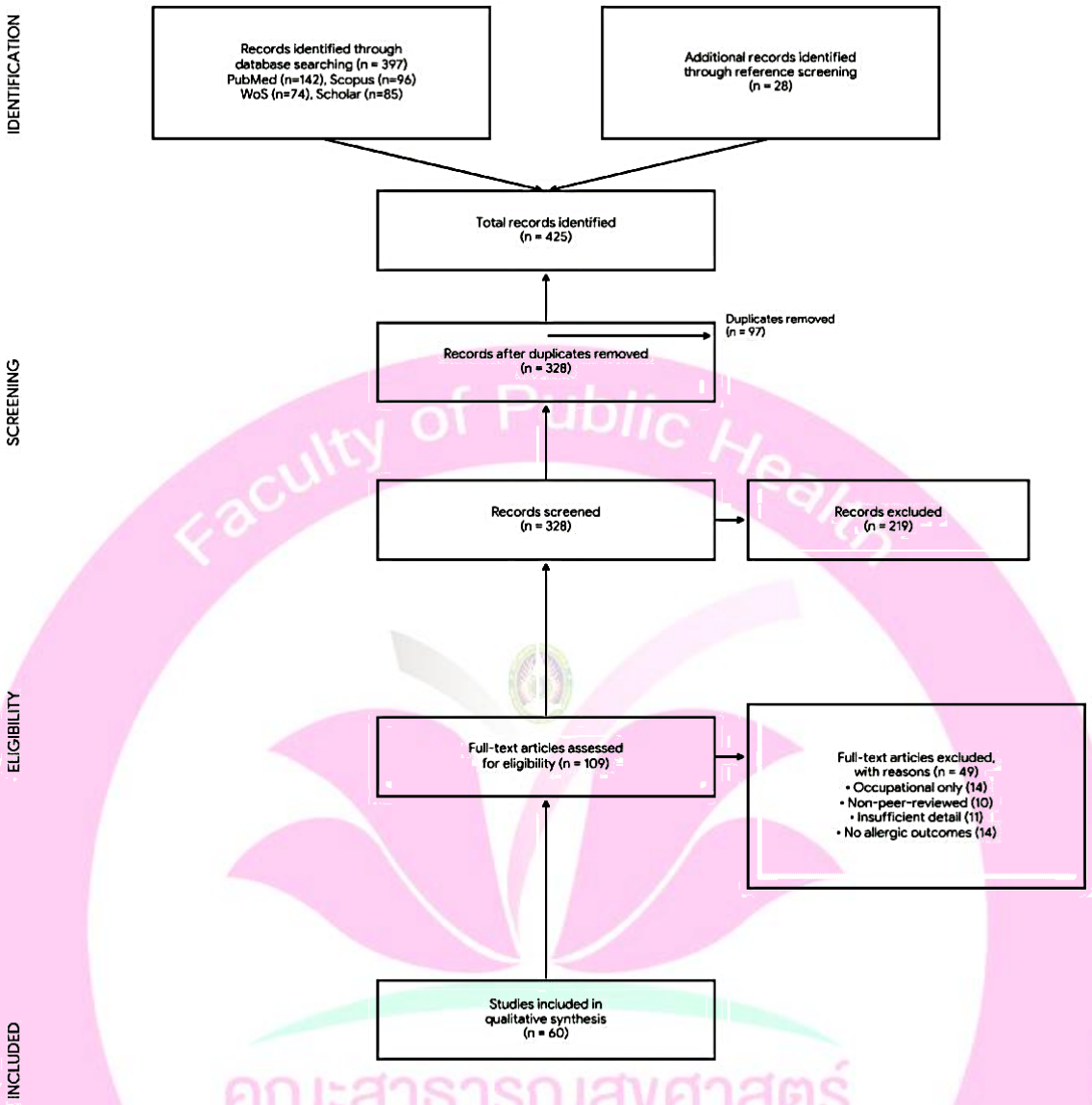


Figure 1 PRISMA flow diagram of study selection

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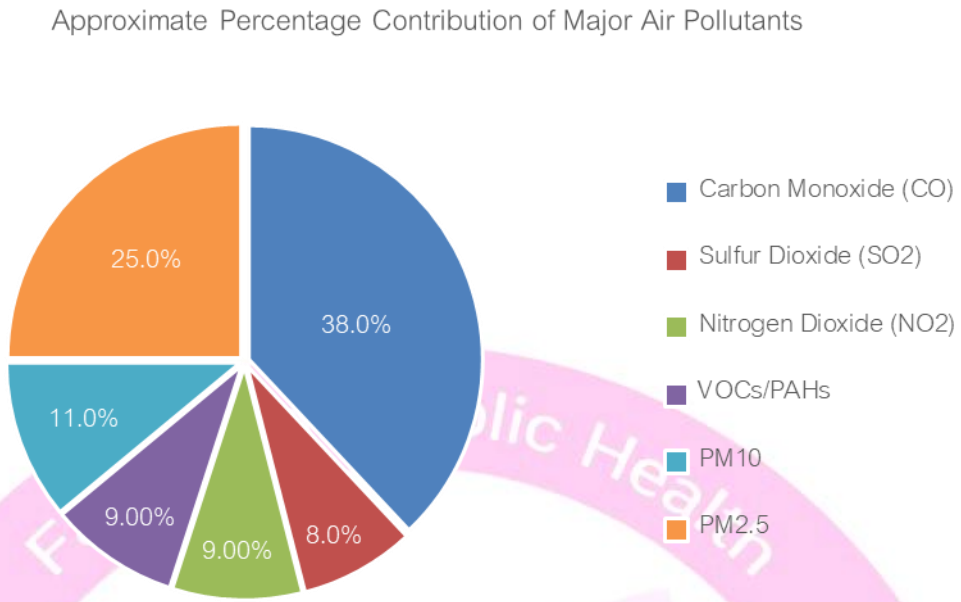


Figure 2 Relative Contribution of Major Ambient Air Pollutants to Overall Exposure Burden

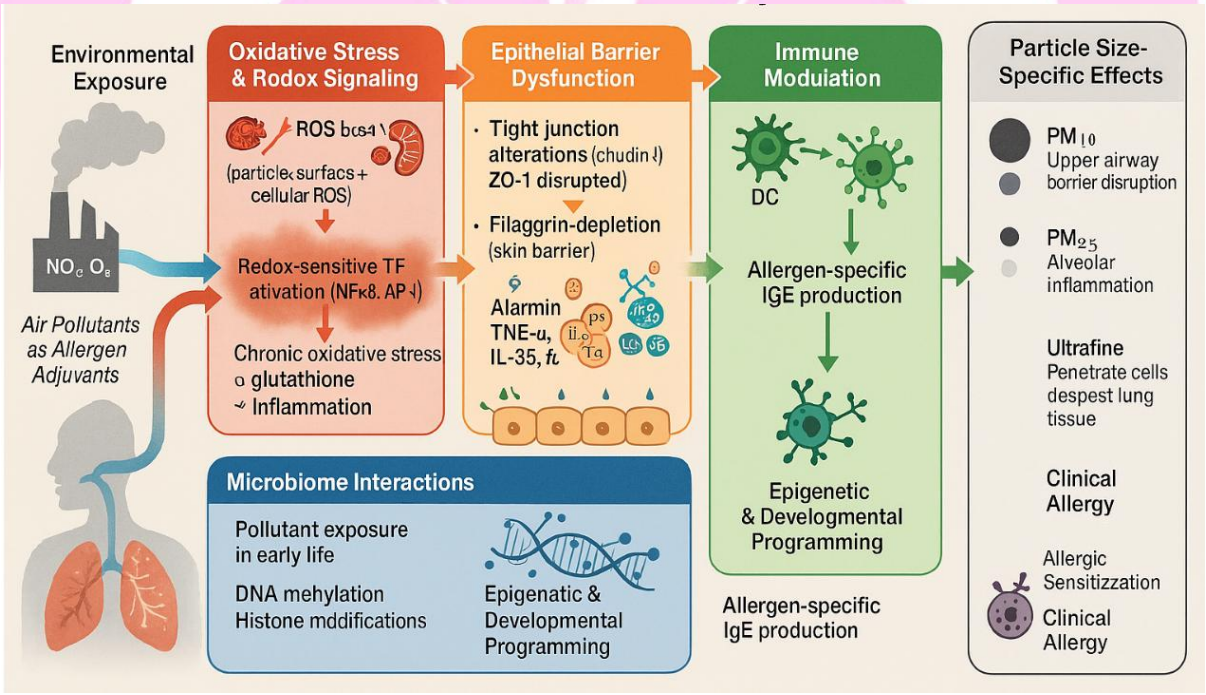


Figure 3 Mechanisms of Allergic Sensitization: Air Pollutants as Immune Adjuvants (Singh et al., 2024)